CLAIMS

(Amendment under the PCT Article 34)

1. An inactive Ca^{2+}/calmodulin-dependent protein kinase IIα (CaMKIIα) knockin nonhuman animal, wherein a CaMKIIα gene of one or both of homologous chromosomes is substituted into an inactive type so that an inactive CaMKIIα is expressed, and thereby a protein kinase activity of the CaMKIIα is specifically impaired while a calmodulin binding capacity of the CaMKIIα and a capacity of multimerizing subunits are maintained.

2.(Amended) The inactive CaMKIIα knockin nonhuman animal according to claim 1, wherein brain's nucleus accumbens has lower neuronal activity as compared to that of a wild-type, while there is no substantial difference in neuronal activities in the cerebral cortex and corpus striatum as compared to those of a wild-type.

3. (Amended) The inactive CaMKIIα knockin nonhuman animal according to claim 2, wherein the inactive CaMKIIα knockin nonhuman animal is produced by a gene targeting method.

4. (Amended) The inactive CaMKIIα knockin nonhuman animal according to claim 3, wherein one or a plurality of amino acid residues in a catalytic domain of the CaMKIIα has been modified.

5. (Amended) The inactive CaMKIIα knockin nonhuman animal according to claim 4, wherein one or a plurality of amino acid residues that is required for binding to ATP has been modified.

6. (Amended) The inactive CaMKIIα knockin nonhuman animal according to claim 5, wherein a lysine residue that is required for binding to ATP has been

modified.

7. (Amended) The inactive CaMKIIα knockin nonhuman animal according to any one of claims 2 to 6, wherein the inactive CaMKIIα knockin nonhuman animal is a rodent animal.

8. (Amended) The inactive CaMKIIα knockin nonhuman animal according to claim 7, wherein the inactive CaMKIIα knockin nonhuman animal is a mouse.

9. An inactive Ca^{2+}/calmodulin-dependent protein kinase IIα (CaMKIIα) knockin cell, wherein a CaMKIIα gene of one or both of homologous chromosomes is substituted into an inactive type so that an inactive CaMKIIα is expressed, and thereby a protein kinase activity of the CaMKIIα is specifically impaired while a calmodulin-binding capacity of the CaMKIIα and a capacity of multimerizing subunits are maintained.